[REVIEW]

How to Use Your Clinical Judgment to Screen for and Diagnose Psychogenic Nonepileptic Seizures without Video Electroencephalogram

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Innov Clin Neurosci. 2011;8(1):36–42

FUNDING: No funding was received for the development of this article.

FINANCIAL DISCLOSURES: The authors have no conflicts of interest relevant to the content of this article.

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KEY WORDS: psychogenic seizures, nonepileptic seizures, pseudoseizures, video electroencephalogram, v-EEG

ABSTRACT

In this article, the authors emphasize the value of proper understanding of the nature of psychogenic nonepileptic seizures in order to reach the right diagnosis based on clinical symptoms and signs. The authors review the literature and provide information regarding epidemiology, etiology and pathogenesis, diagnosis, and features of psychogenic nonepileptic seizures as they compare to epileptic seizures. The authors make suggestions for treatment and provide a clinical diagnostic tool that can aid clinicians in identifying a psychogenic nonepileptic seizure episode.

INTRODUCTION

Psychogenic nonepileptic seizures (PNES) can be deceptive, and a better understanding of their pathology should be the first step in establishing the correct diagnosis of a PNES attack and effective treatment. In the current day practice, video electroencephalogram (v-EEG) is the gold standard in differentiating PNES from epilepsy. However, v-EEG is expensive and time-consuming. Routine inter-ictal EEG findings are generally not considered effective enough to establish an accurate diagnosis of PNES for reasons that will be discussed in this article.

This paper is a literature review that reflects the importance of obtaining detailed history and physical examination of patients presenting with epileptic seizures. By understanding the nature of PNES in patients with disorders masquerading as epilepsy, more effective treatment can be given, even in cases when the

diagnosis of coexistent epileptic seizures (ES) has already been established.

EPIDEMIOLOGY

The prevalence of PNES in the general population ranges from 2 to 33 cases per 100,000 persons. Hence, these seizures are approximately as common as multiple sclerosis and trigeminal neuralgia.[1] Women comprise between 75 and 85 percent of PNES cases.[2] Like other conversion disorders, PNES tends to begin in young adulthood, although the seizures can occur in a wide age range.[2] The mean age for seizure onset in PNES is about 30 years, although seizure onset may also appear during childhood or even in elderly people.[3–10] In a study that analyzed the incidence of PNES in persons aged 15 years or older,[3] the highest age-specific incidence was 4.38/100,000 persons per year for the age range of 25 to 44 years, while it was the lowest in patients over 65 (0.63/100,000 persons per year). Another study[4] found the highest incidence of PNES to be in the age group of 15 to 24 years. Remarkably, the authors in the latter study noted that PNES was not diagnosed in any patient over the age of 55. Diagnostic criteria in these studies required v-EEG monitoring for PNES diagnosis. There are several other reports of PNES in elderly patients.[6–10]

Estimates of the coexistence of ES and PNES vary from 5 percent to more than 60 percent, depending on the study setting and diagnostic criteria.[11] The higher percentages are reported in older studies that were done before use of v–EEG was established, and hence frontal lobe complex partial seizures were missed and labeled as PNES.[12] Recent studies[13,14] using more stringent criteria in diagnosing epilepsy found that only 5 to 10 percent of subjects with nonepileptic seizures had concurrent epileptic seizures. It is estimated that 5 to 10 percent of outpatient epilepsy populations have PNES, compared to 20 to 40 percent of inpatient epilepsy populations (hospitals and specialty epilepsy centers).[11,13]

ETIOLOGY AND PATHOGENESIS

Nonepileptic seizures are classified as physiologic or psychogenic in origin.[11,15] Physiological nonepileptic seizures are less common and may be caused by a variety of conditions that can trigger seizures, such as cardiac dysrhythmias, sudden hypotension, hypoglycemia, sleep disorders, vascular lesions, complicated migraines, panic attacks, and alcohol and drug intoxication or withdrawal. Movement disorders may also be mistaken for nonepileptic seizures.[16]

PNES is defined in modern psychiatry as a conversion disorder, which is classified as one of the somatoform disorders in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*)[17] and is considered a subtype of dissociative disorders in the *International Classification of Disease, Tenth Edition* (*ICD-10*).[18] Conversion disorder is defined as one or more symptoms or signs that affect voluntary motor or sensory function and cannot be explained by neurological or other general medical conditions. Dissociative disorder, on the other hand, is defined as a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment.

From the psychodynamic point of view, PNES functions as a coping mechanism in patients that generally use maladaptive coping strategies to handle stress.[19,20] In these patients, the physical events serve to keep internal stressors out of conscious awareness. Thus, as opposed to factitious disorder or malingering, PNES episodes are not intentional and the patient is not aware of the psychological component of his or her illness. PNES may represent a reinforced behavioral pattern in cognitively impaired patients.[21] Rarely, malingering or factitious disorder may also present with seizures.[21]

Sexual and physical abuse histories have been reported in 24 and 15.5 percent of cases, respectively.[22] Conversion and other psychophysiological disorders in patients with histories of childhood trauma are thought to be the somatic representations of overwhelming and intolerable psychic dilemmas, including recollections, affective suffering, fixed cognitive schema, and severe intrapsychic conflicts.[22–31] Although most adults who were abused as children do not manifest significant abuse-related symptoms, several factors are associated with the appearance of severe symptoms (e.g., PNES). These include high frequency and long duration of abuse, sexual abuse that involved force or penetration, sexual abuse by the child's father, the child's perception of not being believed, lack of support, and even multiple investigatory interviews. PNES patients with history of sexual abuse usually present with a longer diagnostic delay, they are more psychologically unwell, and they are more socially disabled.[31]

The prevalence of PNES is increased in patients with head injuries, learning disabilities, or isolated neuropsychological deficits.[32–35] Commonly associated psychiatric comorbidities include depression, posttraumatic stress disorder (PTSD), other dissociative and somatoform disorders, and personality pathology, especially borderline personality type.[36,37]

CLINICAL DIAGNOSIS

Early diagnosis of PNES is critical. Delays in PNES diagnoses range from a few months to 9 years, with the mean time between the appearance of the clinical manifestations and the establishment of an accurate diagnosis of PNES being 7.2 years.[32] Because of this delay, many patients experience significant morbidity from inappropriate treatment, including adverse effects of antiepileptic drugs and aggressive interventions, such as intubation for pseudostatus epilepticus.[38,39] In addition, an

accurate diagnosis of PNES significantly reduces subsequent healthcare costs.[40] Physicians have an important role in the early diagnosis of these cases through early referral of patients with atypical features to v-EEG monitoring. It is also worth mentioning that the definitive diagnosis of PNES may be therapeutic by itself.[41]

The diagnosis of PNES can be challenging and requires careful evaluation to exclude true epileptic seizures or other medical conditions that cause seizures. Neither epileptic nor nonepileptic seizures should be dealt with as a diagnosis of exclusion—they may coexist. Several clinical features may suggest a diagnosis of PNES,[12,15,32,42–54] which for simplicity can be categorized into four groups: general, pre-ictal, ictal, and post-ictal features.

General features. The general features that can raise one's suspicion of PNES can be recognized during detailed history taking or during review of medical records. High seizure frequency with multiple emergency room visits could be the first clue. PNES can be associated with multiple other psychiatric disorders, including depression, PTSD, other dissociative and somatoform disorders, and personality pathology, especially borderline personality type. History of sexual or physical abuse is important. The negative history of injury or loss of control over bladder or bowel during seizure episodes is also important. Patients with PNES generally lack response to treatment with antiepileptic drugs or have a paradoxical increase in seizures with antiepileptic drug treatment.[15,32,42]

Pre-ictal features. Several pre-ictal features of PNES can be identified. Pre-ictal pseudo sleep may be present,[43] which is a state that resembles normal sleep by behavioral criteria alone (body motionless, eyes closed), while EEG shows evidence of wakefulness (alpha rhythm, active electromyogram [EMG], and rapid eye movement). This feature has a sensitivity of 56 percent and specificity of 100 pecent for pseudoseizure. The onset of the seizure episodes is often gradual and witnessed, and at times begins with stress or with auditory or visual stimuli.[32,42] An emotional or situational trigger can often be appreciated. Seizure may also be provoked by suggestion or by a provocative technique, which may even precipitate a pseudostatus epilepticus.[44,45]

Ictal features. The ictal features of PNES are perhaps the easiest to recognize, as they can often be observed by the clinician.[32,42,46,47] The contractions are usually asynchronous, with nonstereotypical movements that change during the course of the episode. There is absence of the rapid contractions and slow relaxation pattern of true epileptic clonic seizures. Patients having an episode of PNES may exhibit side-to-side head movements. They may also close their eyes forcefully during seizure and resist attempts by the clinician to open their eyelids.[48] However, the observer may be able to modify the patient's motor activity through suggestive comments. Ictal vocalizations (e.g., crying, weeping, and yelling) have been reported.[49-51] In a study of the incidence of ictal stuttering,[51] it was found to be present in 8.5 percent of 117 adult patients with PNES, but was not observed in a consecutive series of 113 adults with epileptic seizures). Another common feature is avoidance or guarding behavior during the seizures. For example, a patient having an episode of PNES usually will not lose balance and fall on the ground if an episode starts while sitting on a chair. Tongue biting, if it occurs, is usually at the tip of the tongue (not the sides as it usually is in epileptic seizures). Patients during a pseudoseizure episode usually maintain control over their bladder and bowel functions. Autonomic changes can occur. Gowers[52] described laryngospasm, large pupils, and decreased sensitivity of the conjunctivae due to increased stress-related sympathetic discharge associated with psychogenic seizures. Light reflex should be intact. Touching the cornea with a cotton swab during seizure episode could help differentiate PNES from epilepsy, as the patient with PNES will likely not allow such a test to be administered due to fear of injury or pain. Finally, PNES usually has a more prolonged course than an epileptic episode (epileptic seizures usually last 2–3 minutes, while PNES can last several minutes to hours).

Post-ictal features. The post-ictal features can also be easy to recognize. A shallow, irregular, and quiet post-ictal breathing pattern that lasts for short duration (about 1 minute) is very suggestive of PNES.[53] The ability of the patient to recall the details of a generalized seizure episode, which requires involvement of both cerebral hemispheres and thus suggesting a loss of consciousness did not occur, contributes to a diagnosis of PNES. Absence of post-ictal symptoms, such as confusion, headache, and fatigue, is a strong negative feature.[54]

Based on the clinical features discussed above, we have constructed a clinical tool we use to screen patients during and after seizure episodes for possible PNES (Table 1).

INVESTIGATIONS

Lab findings associated with an epileptic episode may include elevated serum levels of prolactin, creatine kinase (with a delay of at least 3 hours and a peak concentration after more than 36 hours) and ammonia, and elevated white blood cell count.[55–57] PNES is typically not associated with such changes.

Ambulatory EEG is not considered a reliable tool in diagnosing PNES.[42,58] EEG findings in epilepsy include post-ictal slowing and inter-ictal spikes.[59] Although PNES is essentially, and by definition, not accompanied by the abnormal electrical discharges associated with epilepsy, there is a possibility of some patients having

either focal or generalized slowing due to an independent, organic cerebral pathology. Moreover, inter-ictal epileptiform discharges have been reported on rare occasions in patients without epilepsy, as in the case of inherited EEG patterns, hindering their diagnostic value. Commonly, patients with epileptic seizures may have false-negative EEG results, and, therefore, could be misdiagnosed with PNES, especially in the case of frontal lobe complex partial seizures, as those patients often have no inter-ictal epileptiform abnormality on their scalp EEGs. The use of additional scalp electrodes, especially in combination with anticonvulsant withdrawal, can reduce the number of false negatives during ictal recording.

The importance of using v-EEG monitoring was recently underscored in a study[60] that evaluated patients referred to an inpatient v-EEG monitoring unit for characterization of their seizures. The study found an astonishing 24 percent of the subjects to have been misdiagnosed. Twenty-two patients previously diagnosed with epilepsy were found to have nonepileptic seizures, and four patients previously diagnosed with nonepileptic seizures were given a definitive diagnosis of epilepsy.

Brain magnetic resonance imaging (MRI) evidence of mesial temporal sclerosis as well as hippocampal sclerosis can be seen with PNES,[61] which is often identified as a cause of temporal lobe epilepsy. Clinicians must remember that those MRI findings are only of value when interpreted in the proper clinical context.

MANAGEMENT AND PROGNOSIS

Patient education is critical. The neurologist should continue to monitor the patient with the psychiatrist or psychologist. It is common for neurologists to transfer the complete care of PNES patients to a psychiatrist immediately following disclosure of the diagnosis. Once the diagnosis is made, most PNES patients are taken off of antiepileptic medications by the

TABLE 1. Screening tool to assist in distinguishing between psychogenic nonepileptic seizures and epileptic seizures

OBSERVED	EPILEPTIC FEATURES	NONEPILEPTIC FEATURES
A. Ictal Stage		
Head movements	Fixed	Side to side
Eyes		
Eyelids	Open	Closed
Light reflex	Absent	Present
Corneal touch	No response	Guarding response
Mouth		
Vocalizations	No	Yes
Tongue injury	May bite the side(s)	May bite the tip
Limb movements		
Synchronous	Yes	No
Continuous	Yes	No (with pauses)
Typical	Yes (tonic, clonic, or both)	No
Duration	2–3 minutes	More than 3 minutes
B. Post-ictal Stage		
Breathing		
Deep	Yes	No
Regular	Yes	No
Loud	Yes (with snoring)	No
Duration	5 minutes	1 minute
Recall of episode events	No	Yes
Confusion	Yes	No
Headache	Yes	No
Fatigue	Yes	No

neurologist and care is transferred to psychiatrists. Unfortunately this disrupts the rapport neurologists have with the patients and may have a negative impact on the outcome of PNES. Neurologists should continue to follow patients diagnosed with PNES until the psychopathology is managed and the episodes subside. In rare cases of coexistent epilepsy, seizures may manifest in this interim period, especially off antiepileptic medications. Moreover, premature discharge to psychiatry often contributes to patients' resistance to accept the diagnosis of a psychogenic process. Patients characterized by their denial of any stressors or other psychological problems may refuse initially to seek psychiatric treatment. Patients should be included in the decision-making process of when to stop their visits to the neurologist.[19,62]

The duration of illness is probably the most important prognostic factor in PNES; the longer the episodes continue to occur, the less likely they are to come under control. Outcomes are generally better in children and adolescents than in adults. This may be because the duration of illness is shorter and the psychopathology or stressors are different in pediatric patients than in adults. Other predictors of good outcome include the following: mild psychiatric history, identifiable acute psychological trauma, living independently, and absence of concomitant epilepsy. History of abuse (physical, sexual or emotional), personality disorders, recurrent major depression, dissociative and somatoform disorders, as well as denial of any stressors or psychosocial problems are associated with increased risk of recurrence of PNES.[19,62–64]

The first step in effective treatment of PNES is building a strong therapeutic alliance. Up until diagnosis is confirmed and an appropriate psychiatric referral is made, patients with PNES commonly face lack of interest or disbelief in their complaints and suffering. Therapy should, therefore, start with respect for the patient's symptoms and an acknowledgment of their validity. Reassurance, although seemingly appropriate, should be timed and quantified based on an adequacy of data and strength of the therapeutic alliance.[65]

Psychotherapy can include individual or group therapy, insight-oriented therapy, behavioral techniques, hypnosis, physical therapy, and biofeedback and relaxation training. Overall, cognitive behavioral therapy (CBT) has shown the highest efficacy in treatment of somatoform disorders, including PNES. Behavioral interventions should focus on improving self-esteem, increasing the capacity to express emotions, and build the ability to communicate comfortably with others. Psychological interpretations or explanations should be avoided in the beginning of therapy. Confronting the patient that he or she may be malingering is very counterproductive.[66-69]

Medication management should be very restricted and mainly used to relieve the symptoms of comorbid anxiety or depression.[70]

SUMMARY

PNES can be a devastating illness with significant burden on the patient's health as well as on the community when considering the healthcare costs. Several studies have been conducted to investigate the epidemiology and etiology of PNES. Advances in diagnostic techniques and development of v-EEG now allow for a narrower margin of error when diagnosing PNES. However, we believe a skilled clinician can make a diagnosis of PNES on a clinical basis. We have developed a clinical guide we use to screen patients during and after seizure episodes for possible PNES. Unfortunately, the rich number of studies done on diagnosing PNES is not met with an equal effort to study therapeutic modalities for the illness, and this area deserves more attention from clinical investigators.

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